

The Effect of PRRS Virus Outbreak on Genetic Parameters of Reproductive Performance in Pigs

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Summary and Implications

PRRSV is a significant economic problem for producers in the US and around the world. The genetic basis for reproductive performance in sow herds has been poorly understood. The current analyses shows:

- S/P ratio was lowly heritable (0.17)
- S/P ratio was not highly correlated to reproductive performance during a PRRS outbreak
- Genetic correlations between different outbreak phases ranged between -0.45 to 0.99

Introduction

Porcine reproductive and respiratory syndrome virus (PRRSV) affects both breeding and growing pigs. Out of the estimated total \$664 million cost of PRRS, 45.5% (\$302 M) is due to losses in the breeding herds¹. Despite this impact, limited research is available on the genetics of reproductive performance under a PRRS outbreak for a number of reasons. The most limiting may be the biosecurity in pedigreed populations, leading to a limited number of outbreaks in these herds, whereas most commercial herd are not pedigreed and, therefore, not available for genetic studies.

Materials and Methods

Reproductive data: Data on 2499 Yorkshire (York) and 2732 Landrace (LR) litters were collected from August 2014 to January 2016. Reproductive traits included total number born (TNB), number born alive (NBA), number weaned (NW), number stillborn (NSB), number born mummified (NBM), and total number born dead (NBD, stillborn + mummified). Mortality traits were log transformed for genetic analyses.

Antibody: A PRRS outbreak (1-7-4 strain) occurred in the middle of the 2015 year on three neighboring barns with pedigreed purebred lines. Three weeks after the initial outbreak, all sows in each barn were inoculated with the same strain. Blood samples were taken approximately 45 days after inoculation over four days. Antibody level against the nucleocapsid (N) protein (conserved) was quantified

with a fluorescent microbead assay. Sample-to-positive ratio (S/P) was used as the measure of antibody level. SP ratio is defined as the (sample mean – negative control mean)/(positive control mean – negative control mean).

Analysis: Each trait was split into phases for multivariate analyses by using a mixed model with barn-year-week as a random effect. Three phases: prior, PRRS, and post outbreak were split by visual appraisal of Figure 2.

Genetic parameters were estimated for the same trait between different reproductive phases and between antibody level and reproductive traits. The model for litter size traits included breed, barn, parity, year-month of farrowing, the rolling average of the trait as a covariate, and a random animal genetic effect utilizing the pedigree. The model for antibody level included assay plate, breed, barn, date of collection, and parity, with a random animal genetic effect utilizing the pedigree.

Results and Discussion

30-day rolling averages were calculated for each barn and breed combination. Figure 1 shows all rolling averages for both breeds and all three farms. TNB was affected a few months after the initial outbreak due to the gestation length of the sow. Figure 2 shows the standardized effects for barn-year-week. All traits, except TNB, were affected immediately by the outbreak, with the exception of NSB, which did not show the same secondary response to the inoculation.

Heritability estimates for the litter size mortality traits increased during the outbreak (by 0.01 to 0.08), but decreased for NBA (0.12 to 0.05). S/P ratio had a heritability of 0.17 during the outbreak. TNB did not decrease in heritability until the post phase (from 0.10 prior-to-outbreak to 0.05 post-outbreak).

Genetic correlations of the same trait between the prior and PRRS phases ranged from -0.48 (NBM) to 0.99 (NSB). NBA had a genetic correlation of 0.93 between prior and PRRS phases. TNB had the weakest genetic correlation between prior and post phases at 0.19. Genetic correlations for the same trait between the PRRS and post phases ranged from -0.45 (NBM) to 0.96 (NSB). NBA had the weakest correlation between the PRRS and post phases (0.37).

Genetic correlations of S/P ratio with reproductive performance during the outbreak were low to moderate in general (-0.37 to 0.41). The strongest genetic correlation was for NSB (-0.41). It was expected that mortality traits would be negatively correlated with S/P ratio and that TNB/NBA would be positively correlated, indicating a higher S/P ratio would lead to more TNB/NBA and less incidences of mortality. Genetic correlations from this study

were trending in the expected direction, however they were generally weak.

One previous genetic study of a PRRS outbreak in a sow barn exists with a pedigreed multiplier study². The analysis presented here did not agree very closely with the results of that study, where they found a much higher heritability for S/P ratio and stronger genetic correlations of S/P ratio with reproductive performance. More investigation may be needed into how the quantification of antibody levels differed between studies.

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References

- ¹ Holtkamp et al. (2013) J. Swine Health Prod. 21(2):72-84
- ² Serao et al. (2014) J. Anim. Sci. 92:2905-2921

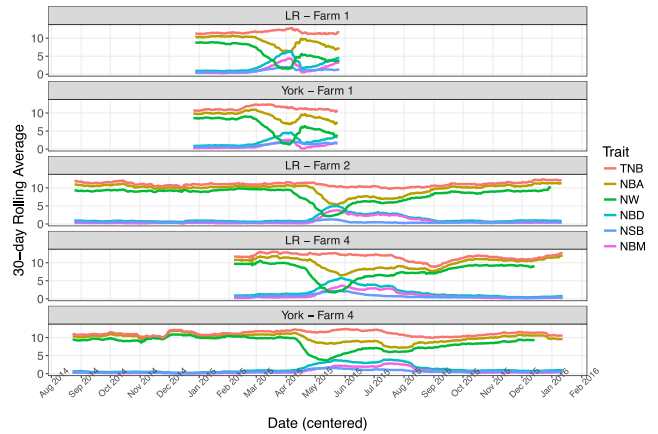


Figure 1. 30-day rolling averages for reproductive performance in both Yorkshire and Landrace in all three barns.

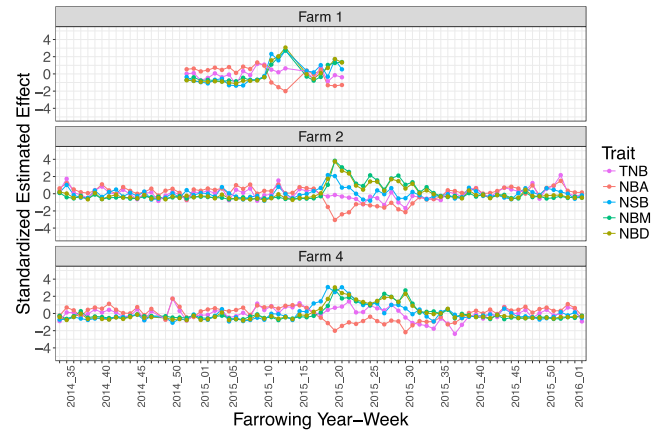


Figure 2. Standardized random barn-year-week estimates from the mixed model for the five traits analyzed in three barns.